**NEMATODES**

***Trichinella spiralis*:** This is one of the smallest nematodes infecting man. The adult worms inhabit the small intestine (duodenum, jejune) of man, pig and rat. The males measure about 1.5 mm long by 40 microns in breadth; thus they are difficult to see with the naked eye. The female is about 3.5 mm in length and 60 microns in breadth. The oesophagus is long and occupies about one-third to one-half of the body length. The anus is terminal in both sexes. The males have no copulatory spicules but a pair of small conical papillae (claspers) is present and they are useful to hold on to the female during mating. These cover the cloaca (anus) on each side. The vulva of the female opens near the middle of the oesophageal region. The anterior half of the body is thin and pointed, well adapted for burrowing into the mucosal epithelium.



Life cycle of *Trichnella spiralis* (from Parasite image library of CDC, USA)

The females are viviparous. The life span of the adult worm is very short. The male dies soon after inseminating the female and the female dies in about 16 weeks after discharging the larva. The larva measures about 80 μm in length by 7-8 μm in diameter. The larva in the cyst is coiled; hence, the species name *spiralis*.Unlike most other nematodes of medical importance, there is no development outside the body of the host. The same host acts both as the primary and the intermediate host. Despite this, two hosts are required to complete the life cycle for the preservation of the species. Man is the end of the species. The continuance of the species is maintained by other animals. The larvae discharged by the female in the mucosa are carried in the lymph stream to the thoracic duct, to the venous system, to the heart, to the lungs, back to the heart and then into the systematic circulation and are distributed to all parts of the body. While they die in most situations, they grow and develop in the striated skeletal (voluntary) muscles. The most heavily parasitized muscles are the diaphragm, tongue, throat, pectoral girdle, cervical, intercostals, jaw and extra-ocular muscles because cysts develop preferentially in muscles relatively poor in glycogen. Cysts are more abundant near the sites of attachment of muscles to tendons and bones than in other parts. In the muscle cell, th0e larvae undergo encystment in a coiled form which is the infective stage.

A muscle cell carrying larva of *T. spiralis* is known *Nurse Cell*. Encysted larvae may remain infective for many years but usually they die and are calcified after about 7 months. Infection is acquired by ingestion of raw flesh of the animal containing viable encysted larva. Man acquires infection by ingesting of raw or undercooked pork containing viable encysted larvae. The cyst wall is dissolved by the action of digestive enzymes; the larvae are set free and within three days moult four times, develop into adults and become sexually matured in 5 days.

**Pathogenicity:** Most infections are subclinical. The minimum of ingested larvae required to produce symptoms is about 100 and a fatal dose is estimated to be 300,000. The pathological changes and symptomatology are divided into three successive stages. (a) Adult female worms

present in the intestine cause gastrointestinal disturbances; (b) Migrating larvae cause various allergic manifestation such as fever, oedema of the face, eosinophilia, and (c) Encysted larvae in the skeletal muscles cause muscular pain.

***Stage of Intestinal invasion* (Enteric phase) *-***The phase occur within the first week after ingestion of infected meat, during the intestinal phase; this phase is associated with the development of larvae into adult. For invading of larvae and adult worms, the wall of intestine is damaged which lead to inflammation of duodenal and jejunal mucosa. Gastrointestinal signs and symptoms may be the first evidence of infection, including fever, disgusting, vomiting, abdominal discomfort, diarrhea etc. Respiratory difficulties may occur, and red blotches erupt on the skin in some cases. This period usually terminates with facial edema and fever five to seven days after the first symptoms.

***Migratory phase or Stage of Muscle invasion-*** This phase, beginning about 7 to 9 days after exposure, is associated withpenetration, deposition and encapsulation of the newborm larvae into muscle cells, initiating a strong inflammatory response. During migration newborn juveniles damage blood vessels, resulting in localized edema, particularly in the face and hands. Following this, muscle swelling, tenderness, pain on movement, and fever usually develop. Wandering juveniles may also cause pneumonia, pleurisy, encephalitis, meningitis, nephritis, deafness, peritonitis, brain or eye damage, and subconjunctival or sublingual hemorrhage. Death resulting from myocarditis (inflammation of heart muscle) may occur at this stage. Although juveniles do not stay in the heart, they migrate through its muscle, causing local areas of necrosis and infiltration of leukocytes.

***Stage of Encapsulation of Encystation phase*.** Encystation occurs only in striated muscle while in other tissues, they degenerate and are absorbed. The formation of cyst results in the stimulationof larvae and tissue reparation. With encystation, the inflammation disappear gradually, the clinicalmanifestation become light, but the muscular pain can still last for months. Eventually all symptoms subside and finally the larvae calcify. The calcified cysts can be seen grossly in exposed muscle fibres.

**Treatment:** Thiabendazole, Mebendazole and Albendazole are drugs of choice.

**Control**

* Properly cooking pork or freezing it at -200 C for 3 days kill the larvae in the pork. Smoking, curing or drying of meet are not dependable methods for killing the larvae.
* Regular inspection of meat, avoidance of eating raw or undercooked pork and meat of other wild animals will prevent transmission of infection to man.
* Preventing pigs from eating raw meat and offal.
* Extermination of rats from pig farms limits the spread of infection

***Strongyloides stercoralis*:** This is also called threadworm. It is the smallest nematode known to cause infection in man. The adult worm is found in the human intestine embedded in the mucosa of the duodenum. Only the female worms are seen in the intestine. It was believed that they are parthenogenetic and can produce offspring without being fertilized by the male. But it has been established that parasitic males do exist. The males are shorter and broader than the female. They are not seen in human infections because they do not invade the intestinal wall and so are eliminated from the bowels soon after the females begin to oviposit.

The female worm is about 2.5 mm and 0.05 mm in breath. It has a cylindrical oesophagus occupying the anterior third of the body and the intestines in the posterior two-thirds opening through the anus situated ventrally a little in front of the pointed tail tip. Paired uteri lead to the vulva situated at the junction of the middle and posterior thirds of the body. The caudal extremity of the female is pointed.

In the gravid female, the uteri contain thin-walled transparent ovoid eggs which have larvae ready to hatch. The worm is ovoviviparous. The eggs laid in the mucosa hatch immediately, releasing rhabditiform (first stage) larvae, with a relatively short muscular oesophagus ending in an enlarged bulb. They bore their way out of the mucous membrane into the lumen from where they are passed in the faeces and may undergo two types of development in the moist, warm soil namely, Direct and Indirect development.

In the direct development, the rhabditiform larvae metamorphose into filariform larvae, and each rhabditiform larva gives rise to one filariform larva. In indirect development, the rhabditiform larvae develop into free-living adult male and females in the soil. After insemination by the male, the female lays a second batch of rhabditiform larvae which are identical to those produced by the parasitic females. The rhabditiform larvae may repeat the free-living cycle or develop into the filariform larvae which infect human and initiate the parasitic phase. Filariform larvae are the infective form of the parasite. The worm passes its life cycle in one host, man. No intermediate host is required. Man acquires infection by walking barefooted on the faecally contaminated soil. The filariform larvae penetrate the skin coming in contact with soil. On penetration the skin, the larvae enter the cutaneous lymphatics or blood vessels and are carried along the venous circulation to the right side of the heart and to the lungs. Here they escape from pulmonary capillaries into the alveoli, migrate up the respiratory tract to the pharynx and are swallowed, reaching their final destination, the duodenum and jejunum, where they burrow into the mucosa. There they mature and start laying eggs.

The worm also has a cycle of autoinfection. Here the rhabditiform larvae mature into the infective third stage larva during their passage down the gut. These filariform larvae cause re-infection by piercing the perianal and perineal skin during defeacation. They ultimately enter the lymphatics or venules and are carried to the right heart and the lungs to complete the life cycle as above without leaving the host and going through a soil phase again. This ability to cause autoinfection explains the persistence of the infection in patients for long periods (30-40 years), after leaving the endemic areas.

**Pathogenicity:** Generally benign and asymptomatic. In symptomatic cases, the following lesions may be seen. The overwhelming severe disease seen in the immunno-compromised is known as hyperinfection.

*Cutaneous Lesions*:- There may be a dermatitis, with erythema and the itching at the site of penetration of the filariform larvae, particularly involving perianal skin and buttocks are common symptoms.

*Pulmonary Lesion*:- When the larvae of *S. stercoralis* migrates through lungs, they break out of the pulmonary capillaries into the alveoli leading to haemorrhages in the lung alveoli and brochopneunomia. These areas are often infiltrated with eosinophils. Occasionally, larvae may lodge in the brochial epithelium and develop to maturity there.

*Intestinal Lesions*: - The symptoms may resemble those of peptic ulcer or of mal-absorption. Mucus diarrhoea is often present. In heavy infection, the mucosa may be honey combed with the worm and these may be extensive sloughing, causing dysenteric stools.

*Hyperinfection*:- People with hyper-infection generally experience a worsening in abdominal symptoms, often paralytic ileum, gastrointestinal bleeding and perforation. The increase in number of worms migrating through the lungs results in wheezing, dyspnoea and pulmonary haemorrhage. During this stage, filariform larvae may enter into arterial circulation and lodge in various organs e. g. lymph nodes, endocardium, pancreas, liver, kidneys and brain. The signs and symptoms will depend upon the organ affected.

**Treatment:** Thiabendazole, Mebendazole and Ivermectin are effective drugs.

**Control**

* Proper disposal of human waste
* Avoidance of contact with faecally contaminated soil.
* Treatment of all diagnosed cases.

***Ancylostoma duodenale*:** The adult worms live in the small intestine of man, mostly in the jejunum, less often in the duodenum and infrequently in the ileum. They are relatively stout cylindrical worms. The body is curved with the dorsal aspect concave and the ventral aspect convex. The anterior end is somewhat constricted and bent dorsally, hence the name “hookworm.” The mouth is not at the tip but directly dorsally. The prominent buccal capsule, reinforced with a hard chitin-like substance carries two pairs of hook-like teeth ventrally and a dental plate with a median cleft dorsally. The posterior end of the male is expanded into a copulatory bursa supported by fleshy rays. The pattern of the rays helps in distinguishing between different species. The cloaca into which the rectum and genital canal is open is situated within the bursa. There are two long retractile bristle-like copulating spicules, the tips of which project from the bursa. The female is large. Its hind end is conoid, with a sub-terminal anus situated ventrally. The vulva opens ventrally at the junction of the middle and posterior third. The vagina leads to two intricately coiled ovarian tubes which occupy the hind and middle parts of the worms. The copulating bursa is used for attachment to the female during copulation. The eggs are oval or elliptical, colourless, not bile stained, with a thin transparent hyaline shell membrane. When released by the worm in the intestine, the eggs contains an unsegmented ovum. During its passage down the intestine, the ovum develops. Man is the only host. Eggs freshly passed in faeces are not infective for humans. When deposited in the soil, the embryo develops inside the eggs. In the warm, shady and moist, rhabditiform larvae hatch out from the eggs in 2 days. They feed on bacteria and other organic matter in the soil, grow in size and moult twice and produce the third larval stage; the filariform larva which is the infective stage; the filariform larvae are non-feeding and show negative geotropism. They can live in the soil for about 5 weeks in the soil depending on the temperature and water content of the soil. When a person walks barefooted on soil containing the filariform larvae, they penetrate the skin and enter the subcutaneous tissue through the skin between the toes, the dorsum of the foot and the medial aspect of the sole. In farmer workers and miners, they may penetrate the skin of the hands. Rarely entry may be through skin on the other parts of the body. In the subcutaneous tissue the larvae enter the venules and are carried in circulation to the right heart and to the lungs (pulmonary capillaries); where they break through the capillary walls and enter into the alveolar spaces. From alveoli, they migrate up the bronchi, trachae and larynx, crawl over epiglottis to the pharynx are swallowed. During migration, they undergo the third moulting in the oesophagus and the fourth moulting in the small intestine (jejunum); develop the buccal capsule and grow into adults. They attach themselves to the mucous membrane of small intestine by means of their mouth parts and in about 6 weeks the male fertilizes the female and the life cycle is repeated. Transmission can take place by oral, trans-mammary and transplacental transmission.

**Pathogenecity:** This is due to larvae or adult worm. When the larvae enter the skin, they cause severe local itching which may be followed by scratching and secondary bacterial infection-ground “itch” or ancylostma dermatitis. When the larvae break through the pulmonary capillaries and enter the alveoli, they may lead to bronchitis and bronchopneumonia- pulmonary lesions. Adult worm bring about anaemic conditions by ingesting blood and also their secretions contain anticoagulant which encourages bleeding to continue for sometimes from abandoned sites of attachment of the worm. The degree of anaemia depends on the number of worms, body iron store and dietary iron. Hookworm anaemia leads to severe lassitude and dullness, affecting the working and learning capacities of patients and cause mental retardation in children. Hookworm infection may cause intestinal syndrome resembling peptic ulcer, with epigastic pain, dyspepsia and vomiting. There may be diarrhoea, the stool being reddish or black.

**Treatment:** Mebendazole, pyrantel pamoate or albendazole may be used. Thiabendazole and albendazole are used for the treatment of cutaneous larva migrants. In relief of anaemia, oral iron is effective but in severe cases, a preliminary packed cell transfusion may be needed.

**Control**

* Prevention of soil pollution with faeces and proper disposal of night soil
* The use of footwear prevents entry of larvae through the skin of the foot.
* Gloves give similar protection to the hands of farm workers.
* Treatment of patients and carriers limits the source of infection.

***Ascaris lumbricoides*:** This is the largest nematode parasite in the human intestine; particularly the jejunum. It is a large cylindrical worm, with tapering ends; the anterior end has three finely denticulated lips, one dorsal and two ventro-lateral. The cuticle is thick and imparts some rigidity to the worm. The lateral line systems, within which the excretory canal is situated, can be seen externally as a white stripe running along the entire length of the worm. The digestive and respiratory organs of the worm float inside the body cavity possessing a toxic fluid known as ***ascaron***. Allergic reactions seen in infected individuals are due to this toxin.

The male measures 15 to 30 cm in length and 2 to 4 mm in thickness. Its posterior end is curved ventrally to form a hook and carries two copulatory spicules. The female is larger, 20 to 40 cm long and 3 to 6 mm thick. Its posterior extremity is straight and conical. The vulva is situated mid-ventrally, near the junction of the anterior and middle thirds of the body. A distinct groove is often seen surrounding the worm at the level of the vulvar opening. This is called the vulvar waist or genital girdle and is believed to facilitate mating. Two types of eggs are passed by the worms. The fertilized eggs, laid by females inseminated by mating with a male, are embryonated and develop into the infective eggs. The uninseminated female also lays eggs, but these are non-embryonated and cannot become infective. These are called unfertilized. The female worms are very prolific, laying up to 200,000 eggs per day. The eggs are passed in faeces and are not infective when passed but they are very resistant to adverse environmental conditions. The fertilized egg embryonated outside the host under suitable environmental conditions, the egg gives rise to a coiled rhabditiform larva in 10 days or longer depending on the temperature. The larva, still within the egg, moults to produce the second stage larva. This is the infective stage. Man becomes infected when he ingests the infective egg through food, water or raw vegetables. When the swallowed eggs reach the duodenum, the larvae hatch out. The rhabditiform larvae penetrate the gut wall, enter the portal vessels and are carried to the liver. They remain in the liver for a few days and eventually they are carried in the blood to the heart and then to the lungs, where they grow and moult twice. They break out of the lung capillaries into the lung alveoli; where they then break out and migrate up the trachea to the pharynx and then pass down the oesophagus to reach the intestine. The larvae moult and develop into adults in the upper part of the small intestine. They become sexually mature and another cycle is repeated.

**Pathogenicity:** Ascariasis is caused by both adult worms and migrating larvae.

The Adult- By robbing the host of its nutrition which leads to malnutrition and night blindnesss due to vitamin A deficiency. This later brings retardation of growth. Adult worms in the intestine may lead to intermittent colicky cramps and loss of appetite. The worms may cause obstruction of the intestinal tract. The worms are restless wanderers. They tend to probe and insinuate themselves into any aperture they find on the way. They may craw out of mouth or may enter the nasal meatus. They can cause respiratory obstruction, appendicitis, obstructive jaundice and acute haemorrhagic pancreatitis. They may perforate the intestinal wall weakened by ulcers or gangrene. The release of toxic body fluid (ascaron) of the adult worm in the body of the patient may lead to various allergic manifestations such as fever, urticaria, angioneurotic oedema, wheezing and conjunctivitis.

The Larvae- The migrating larvae may lead to inflammatory and hypersensitivity reactions in the lungs and other organs such as liver and kidneys. There is formation of granuloma and eosinphilic infiltrates. It leads to fever, cough, dyspnoea, urticarial rash and eosinophilia. The sputum may be blood-tinged and may contain *Ascaris* larvae. This is known as “Loeffler’s Syndrome”

**Treatment:** Safe and effective drugs include pyrantel pamoate, Mebendazole, albendazole, piperazine citrate.

**Control**

* Only treated night soil should be used as manure.
* Proper disposal of human faeces.
* Avoidance of eating raw vegetables and salads.
* Periodic treatment with an effective antihelminthic in communities that lack sanitary facilities.
* Treatment of vegetables and other garden crops with water containing iodine 200 ppm for 15 minutes kills the eggs and larvae of *Ascaris*.

***Wuchereria bancrofti:*** Adult male and female worms reside in the lymph nodes and lymphatic vessels of man. The adults are whitish, translucent, thread-like worms with smooth cuticle and tapering ends. The female is larger than the male. The posterior end of female worm is straight, while that of the male is curved ventrally and contains two spicules of unequal. Males and females remain coiled together usually in the abdominal and inguinal lymphatics and in the testicular tissues. The adults live for many years, probably 10 to 15 years or more.

It passes its life cycle in two hosts. Man is the definitive host and the female mosquito belonging to genera *Culex, Aedes* and *Anopheles* act as intermediate hosts. The worm is ovoviviparous. The male fertilizes female and the gravid female gives birth to microfilariae. The embryo (microfilaria) is released encased in its elongated egg-shell, which persist as a sheath. The microfilaria has a colourless, translucent body with a blunt head and pointed tail. It is actively motile and can move forward and backwards within the sheath which is much longer than the embryo. The microfilariae circulate in the bloodstream and show a nocturnal periodicity in peripheral circulation, being seen in large numbers in peripheral blood only at night between 10 pm and 4 am. This correlates with the night biting habits of the mosquito vector. Periodicity is also related to the sleeping habits of the hosts. If the sleeping habits of the hosts are reversed, over a period, the microfilariae change their periodicity from nocturnal to diurnal. Microfilariae do not multiply or undergo any further development in the human body and if they are not taken up by a female mosquito vector, they die. Their lifespan is about 2 to 3 months. When a mosquito vector takes the microfilariae in with the blood meal and they reach the stomach of the mosquito; they cast off their sheath within 2-6 hrs., penetrate the stomach wall and within 4-7 hrs migrate to the thoracic muscle where they undergo further development. During the next 2 days, they metamorphose into first-stage larva which is a sausage-shaped form with a spiky tail. Within a week, it moults once or twice, increases in size and becomes the second-stage larva. In another week, it develops its internal structures and becomes the elongated third-stage filariform larva. This is actively motile and is the infective larva. It enters the proboscis sheath of the mosquito, awaiting opportunity for infecting humans on whom the mosquito feeds. There is no multiplication of microfilaria in the mosquito; one microfilaria develops into one infective larva only. When a mosquito with infective larvae in its proboscis feeds on a person, the larvae get deposited, usually in pairs, on the skin near the puncture site. The larvae enter through the puncture wound or penetrate the skin by themselves. After penetrating the skin, the third-stage larvae enter the lymphatic vessels and are carried usually to abdominal or inguinal lymph nodes, where they develop into adult forms. There is no multiplication at this stage and only one adult develops from one larva male or female. They become sexually mature in about 6 months, mate and the cycle continues.

**Pathogenicity:** The lymph nodes become enlarged, firm and fibrotic. Mechanical irritation caused by the movement of adult parasite inside the lymphatic system, liberation of metabolites by growing larvae, absorption of toxic products from dead worms and secondary bacterial infection lead to lymphangitis with swelling, redness, and pain. Because of the chronic inflammation, the lymph valves proximal to the worm become damaged and incompetent. Permeability of the walls of the lymphatics increases, which permits the leakages of fluid with high concentration of protein into surrounding tissues. Lymphatic obstruction may result from mechanical blocking of the lumen by dead worms. Repeated leakage of lymph into tissues results first in lymphedema, then to elephantiasis of limbs, breast, penis, scrotum or vulva. In males, hydrocele, orchitis, fumiculitis and epididymitis are common. Patients develops intermittent recurrent fever with headache, malaise, localized pain and tenderness with oedema and erythema above lymph vessels and glands, accompanied by acute lymphangitis and lymphademitis of the groin or axilla.

**Treatment:** Diethylcarbamazine (DEC) is a drug of choice for the treatment of the infection.

**Control:**

* Destruction of vector by spraying residual insecticides such as DDT, malathion etc. into common resting sites.
* A film of oil may be sprayed over water surfaces to kill the larvae.
* Larvivorous fish may be added ino ponds
* Open drains septic tanks. Soakage pits and flood pit latrines should be adequately maintained.
* Detection and treatment of carriers.